Journal of Population Therapeutics & Clinical Pharmacology

**RESEARCH ARTICLE**
**DOI: 10.53555/jptcp.v31i5.6317**

# CLINICOPATHOLOGICAL COMPARISON OF HISTOPATHOLOGY OF PLACENTAE IN GESTATIONAL DIABETES MELLITUS COMPARED WITH THAT OF UNCOMPLICATED PREGNANCY AND PERINATAL OUTCOME IN GDM MOTHERS IN DIFFERENT INTERVENTIONS IN PREGNANCY

**Dr. Punyatoya Punyashree Sethy[1], Dr. Subhra Ghosh[2], Dr. Saumya Nanda[3*], Dr. Mahija Sahu[4]**

[1]Senior Resident, Department Of Obstetrics And Gynaecology, Shri Jagannath Medical College, Puri, Odisha, India.
[2]Professor And Hod, Department Of Obstetrics And Gynaecology, Shri Jagannath Medical College, Puri, Odisha, India.
[3*]Associate Professor, Department Of Obstetrics And Gynaecology, Shri Jagannath Medical College, Puri, Odisha, India.
[4]Professor & Hod, Department Of Obstetrics And Gynaecology, Mkcg Medical College, Berhampur, Odisha, India.

***Corresponding Author:** Dr. Saumya Nanda
*Associate Professor, Department Of Obstetrics And Gynaecology, Shri Jagannath Medical College, Puri, Odisha, India.

**Abstract**

**Background:** This Study Was Conducted To Study The Histopathology Of Placenta Complicated With Gdm With Placenta With Normal Pregnancy And To Compare The Perinatal Outcome In Gdm Cases With Different Treatment Types Of Interventions.

**Methods:** This Was A Hospital-Based Prospective Case Control And Observational Study That Took Place From January 2020 To August 2021 In The Department Of Obstetrics And Gynaecology And Department Of Pathology, Scb Mch, Cuttack, Orissa. The Study Involved 50 Antenatal Mothers Who Were Diagnosed As Gdm In Opd And Labour Room. The Study Was Approved By The Institutional Ethics Committee, And The Participants Provided Written Informed Consent.

**Results:** The Prevalence Of Gdm (P-Value < 0.002) Strongly Correlated With Increasing Age (Mean Age 29.38±3.09 Years, 86%, >25 Yrs.). Every Morphological Characteristic, Including The Number Of Cotyledons (Mean No. 18.72), Central Thickness (Mean 3.06 Cm), And Placental Weight (Mean Weight 646.58 G) (P-Value < 0.001), Was Statistically Significant. Maternal Decidual Vasculopathy (80%), Fibrinoid Necrosis Of Vessels (86%), Villous Edema (80%), Calcification (68%), Thickened Basement Membrane (100%), Syncytial Knot Formation (10%), And Villous Immaturity (84%), Chorangiosis (82%), Villous Fibrosis (82%), Intervillous And Perivillous Fibrin Deposits (82%), Were All Statistically Significant Histopathological Features. The Mode Of Delivery Did Not Differ

Statistically Significantly Between The Groups (Lscs 46%, P-Value = 0.309). The Mean Birth Weight Of 3.50 Kg Demonstrated Statistical Significance, Nonetheless. Macrosomia (42%) And Hypocalcemia (40%) Were Reported To Be The Two Most Common Unfavorable Fetal Outcomes In Newborns With Gdm. Of The Patients With Gdm, 68% Followed A Diet Plan, 22% Received Insulin Along With A Diet Plan, And 10% Had Not Received Any Treatment At All. Both Macrosomia And Hypoglycemia Demonstrated Statistical Significance In All The Gdm Therapy Groups.

**Conclusion:** Gdm Is More Common In Increasing Maternal Age. All Morphological Characters Like Weight Of Placenta, Central Thickness, Number In Cotyledons Except Diameter Of Placenta Are Increased In Gdm Which Is Statistically Significant. All The Histopathological Changes Are Also Statistically Significant. Perinatal Outcome In Unfavorable In Gdm Mothers Who Did Not Receive Any Therapy.

**Keywords:** Clinicopathological Comparison, Histopathology, Placentae, Gestational Diabetes Mellitus, Perinatal Outcome.

## Introduction

The Term "Gestational Diabetes Mellitus" Refers To Glucose Intolerance That Initially Manifests Or Begins During Pregnancy.[1] Globally, The Prevalence Of Gdm (Gestational Diabetes Mellitus) Ranges Between 2-14%.[2] Depending On The Region And Diagnostic Techniques Employed, The Prevalence Of Gdm In India Ranges From 3.8% To 21% In Various Regions.[3] With An Estimated 63 Million Cases Worldwide, India Has The Second-Highest Number Of Diabetics.[4] Studies Also Indicate That Poor Food, Adolescent Obesity, And A Sedentary Lifestyle Are Contributing To The Rising Prevalence Of Dm (Diabetes Mellitus) Among Pregnant Women.[5] During Pregnancy, The Placenta Is An Organ That Forms And Operates Temporarily. It Performs A Variety Of Functions, Such As Transport And Endocrine Functions, And Acts As A Barrier Separating The Circulation Of Blood In The Mother And The Fetus.[6] The Exchange Of Chemicals Between The Mother And The Fetus Is Its Primary Function. The Placenta Is Composed Of Trophoblastic Cells, Connective Tissue, And Blood Arteries. The Chorionic Villi Are The Primary Functional Components Of The Placenta. Within These Villi, Vasculosyncytial Membranes Covering Dilated Fetal Capillaries Divide Fetal Blood From Maternal Blood In The Surrounding Intervillous Area.[7]

Histologically, A Placenta Has A High Number Of Syncytial Knots And Villi. Syncytiotrophoblast Nuclei Congregate In Clusters Within These Knots, Resulting In Thin Cytoplasmic Regions Devoid Of Nuclei Between Them.[8] The Five Layers That Make Up The Diffusion Barrier Between The Circulation Of The Mother And The Fetus Are The Endothelium, Capillary Endothelial Basement Membrane, Trophoblast, And Core Of Supporting Tissue.[9] Any Kind Of Diabetes Mellitus During Pregnancy Can Result In A Wide Range Of Problems In The Placenta.[10] The Glucose Level At The Crucial Stage Of Placental Development Is One Of The Many Variables That Determine The Kind And Degree Of These Alterations.[11] Uncontrolled Diabetes Alters Placental Function, Which Can Lead To Intrauterine Growth Retardation, Macrosomia, Congenital Malformations, And Abnormalities In Fetal Growth And Development.[12] In Diabetic Pregnancies, There Is An Increased Risk Of Polycythemia, Spontaneous Miscarriages, Intrauterine Death, Surgical Deliveries, Birth Trauma, Newborn Respiratory Distress, Neonatal Hypoglycemia, Hypocalcemia, Hyperbilirubinemia, And Type 2 Diabetes In The Progeny. Preeclampsia, Polyhydramnios, Obstructed Labor, Shoulder Dystocia, Cesarean Section, Postpartum Hemorrhage, And Infection Are Among The More Common Maternal Problems.[13] The Placental Structure Has Been Studied Using Classical Histology, Which Has Revealed Changes In The Syncytiotrophoblast, Cytotrophoblast, Trophoblastic Basement Membrane, And Fetal Vasculature To Differing Degrees.[14,15] Since 1950, The Majority Of Authors Have Documented A Relative Placental Immaturity,[16-18] Most Likely As

A Result Of A Large Percentage Of Villi Exhibiting Localized Fibrinoid Necrosis And Stromal Edema.[19-21]

**Aims And Objectives**

- To Study The Effect Of Gdm On Placental Morphology,Histopathology And Perinatal Outcome.
- To Compare The Morphology And Histology Of Gdm And Normal Placenta.
- To Evaluate The Perinatal Outcomes In Neonate Of Gdm With Different Treatment Modalities.

**Materials & Methods**

This Was A Hospital-Based Prospective Case Control And Observational Study That Took Place From January 2020 To August 2021 In The Department Of Obstetrics And Gynecology And Department Of Pathology, Scb Mch, Cuttack, Orissa. The Study Involved 50 Antenatal Mothers Who Visited The Opd And Labor Room And Who Were Screened For Gestational Diabetes During Their First Trimester Visit. The Study Was Approved By The Institutional Ethics Committee, And The Participants Provided Written Informed Consent.

**Inclusion Criteria**

1. Age 20-35 Years
2. Singleton Pregnancies
3. Primi And Multi Gravida
4. Cases: All Placentas Between The Gestational Age Of 36 Completed Weeks To 40 Weeks Of Gestation From Diagnosed Gdm Mothers.
5. Control Group: All Placentas Between 36 Completed Weeks To 40 Weeks Of Gestation From Uncomplicated Pregnancies.
6. Neonatal Outcomes Are Observed For Macrosomia, Hypoglycemia, Hypocalcemia, Hyperbilirubinemia And Polycythemia In Gdm Patients With Different Treatment Modalities.

**Exclusion Criteria**

1. Gestational Diabetes In Association With Hypertension
2. Gestational Diabetes In Association With Any Toxemia In Pregnancies
3. Any Known Illness Or Metabolic Disorders In Pregnant Mothers
4. Multiple Pregnancies And Iugr
5. Prom, Pprom
6. Overt Diabetes

**Statistical Methods**

Statistical Analysis Was Done Using The Student's T-Test Or Chi Square Test. A P-Value<0.05 Was Considered Significant.

**Results**

| | **Cases Mean ± Sd** | **Control Mean ± Sd** | **P-Value** |
|---|---|---|---|
| Age (In Years) | 29.38 ± 3.09 | 25.76 ± 2.07 | <0.001 |
| ***Table 1:*** *Comparison Of Age Between The Groups* | | | |

The Average Age In The Case Group Was 29.38±3.09 Years, Whereas It Is 25.76 ± 2.07 Years In The Control Group. The Age Range Of The Cases Group Was 23–36 Years, Whereas It Was 21–29 Years In The Control Group. The Maternal Age Distribution Revealed That The Case Group Had A Higher Percentage Of Mothers (86%) Who Were Over 25, Compared To 58% In The Control Group. There Is A Statistically Significant Mean Difference (P-Value<0.001).

| Variables | Cases | | Controls | | P-Value |
|---|---|---|---|---|---|
| | Mean | Sd | Mean | Sd | |
| Placental Weight In Grams | 646.58 | 68.43 | 514.59 | 53.43 | <0.001 |
| Central Thickness In Cm | 3.06 | 0.96 | 2.49 | 0.22 | <0.001 |
| Placental Diameter In Cm | 18.31 | 1.77 | 18.10 | 1.76 | 0.574 |
| Number Of Cotyledons | 18.72 | 0.92 | 16.50 | 1.18 | <0.001 |

***Table 2:*** *Comparison Of Placental Factors Between The Study Groups*

The Mean Weight Of The Placenta Is 646.588g In The Diabetic Group Which Is Greater Than The Mean Weight Of The Placenta In Control Group (514.59g) And Is Statistically Significant (P-Value<0.001).

Other Parameters Like Central Thickness Of Placenta, Number Of Cotyledons Were More In Diabetic Group And Statistically Significant (P-Value<0.001). The Mean Placental Diameter Of Cases Was Slightly Larger Than The Control Group, With A Mean Difference Of 0.21 Mm That Was Not Statistically Significant (P-Value = 0.574).

| Features | Cases | | Controls | | P-Value |
|---|---|---|---|---|---|
| | Number | % | Number | % | |
| Villous Immaturity | 42 | 84 | 7 | 14 | <0.001 |
| Chorangiosis | 41 | 82 | 10 | 20 | <0.001 |
| Villous Fibrosis | 41 | 82 | 0 | 0 | <0.001 |
| Intervillous, Peri-Villous Fibrin Deposit | 41 | 82 | 0 | 0 | <0.001 |
| Maternal Decidual Vasculopathy | 40 | 80 | 0 | 0 | <0.001 |
| Fibrinoid Necrosis Of Vessels | 43 | 86 | 0 | 0 | <0.001 |
| Villous Oedema | 40 | 80 | 0 | 0 | <0.001 |
| Calcification | 37 | 68 | 7 | 14 | <0.001 |
| Thickened Basement Membrane | 50 | 100 | 0 | 0 | <0.001 |
| Syncytial Knot Formation | 50 | 100 | 17 | 34 | <0.001 |

***Table 3:*** *Comparison Of Histopathological Features Of The Placenta In The Study Groups*

When The Placentas Of The Study Participants Were Compared For Histopathological Features, We Found That The Majority Of The Diabetic Group's Placentas Had Villous Immaturity (84%), Chorangiosis (82%), Villous Fibrosis (82%), Intervillous And Perivillous Fibrin Deposits (82%), Maternal Decidual Vasculopathy (80%), Fibrinoid Necrosis Of Vessels (86%), Villous Edema (80%), Calcification (68%), Thickened Basement Membrane (10%), And Syncyitial Knot Formation (10%). In Contrast, The Control Group's Placentas Displayed Villous Immaturity (14%), Chorangiosis (20%), Syncytial Knot Formation (34%), And Calcification (14%). There Was No Evidence Of Villous Fibrosis, Thicker Basement Membrane, Maternal Decidual Vasculopathy, Intervillous And Perivillous Fibrin Deposits, Or Villous Edema On Any Placenta In The Control Group. The P-Values For Each Histopathological Feature Indicate A Statistically Significant Difference (P<0.001).

| | Cases | | Controls | | P-Value |
|---|---|---|---|---|---|
| | Mean | Sd | Mean | Sd | |
| Birth Weight | 3.50 | 0.46 | 3.22 | 0.32 | 0.001 |

***Table 4:*** *Comparison Of Birth Weight Between The Study Groups*

The Idm (Infants Of Diabetic Mothers) In The Current Study Weighed 3.50 Kg At Birth, Which Was 3.22 Kg For Controls, With A Mean Difference Of 280 Gm That Was Statistically Significant (P-Value = 0.001).

| Outcome | Diet Plan | Diet With Insulin | No Treatment | P-Value |
|---|---|---|---|---|
| Hypoglycemia | 14 (41.2) | 2 (18.2) | 0 (0) | 0.002 |
| Macrosomia | 13 (38.2) | 5 (45.5) | 3 (60.0) | 0.032 |
| Hypocalcemia | 12 (35.3) | 6 (54.5) | 2 (40.0) | 0.231 |
| Polycythemia | 0 (0) | 1 (9.1) | 2 (40.0) | 0.093 |
| Hyperbilirubinemia | 10 (29.4) | 6 (54.5) | 2 (40.0) | 0.321 |

***Table 5:*** *Comparison Of Fetal Outcomes In Different Treatment Groups Of Gdm*

In This Study, 38.2% Of Neonates Developed Macrosomia In The Diet Plan Group And 45.5% Of Neonates Developed Macrosomia In The Diet Plan + Insulin Received Group, With A Statistically Significant P-Value (0.032). Additionally, 41.2% Of Neonates Developed Hypoglycemia In The Diet Plan Group Compared To 18.2% In The Diet Plan + Insulin Group. In Patients Adhering To A Diet Plan, 35.3% Of Newborns Had Hypocalcemia And 29.4% Had Hyperbilirubinemia. When Newborns Were Born To Mothers Who Had Followed A Diet Plan, We Did Not Find Any Cases Of Polycythemia. Of Mothers Who Got A Diet Plan Plus Insulin Treatment, The Percentage Of Newborns With Hypocalcemia, Polycythemia, And Hyperbilirubinemia Was 54.5%, 9.1%, And 54.5%, Respectively. Compared To The Diet Plan Group And The Diet Plan + Insulin Treatment Group, The Incidence Of Macrosomia (60%), Polycythemia (40%), Hyperbilirubinemia (40%) And Hypoglycemia (40%) Was Higher In Individuals Who Had Not Received Any Treatment.

**Discussion**

The Participants In This Study Had An Average Age Of 27.57 ± 3.19 Years. The Mean Age Of The Case Group Was 29.38±3.09 Years, While The Control Group's Mean Age Was 26.76±2.07 Years. The Age Range Of The Cases Group Was 23–35 Years, Whereas The Control Group's Age Spanned From 21–29 Years. There Was A Statistically Significant Mean Difference (P Value<0. It Was Found That 86% Of Those In The Case Group Were Over 25, Compared To Only 58% In The Control Group. Statistical Significance Was Also Shown By This Difference (P Value = 0.002). According To Our Research, There Is A Positive Correlation Between Maternal Age And The Chance Of Developing Diabetes During Pregnancy, Which Is Consistent With Other Studies. [22,23] When The Study Participants' Gravidity And Parity Were Compared, It Was Found That 26% Of The Case Group Subjects Were Primigravida, 34% Of The Control Group Subjects Were Primigravida, And 62% Of The Case Group Subjects Were Multiparous, Compared To 50% Of The Control Group Subjects Who Were Multiparous. With A P-Value Of 0.313, This Difference Was Not Statistically Significant.

The Case And Control Groups Do Not Differ Significantly In Terms Of Gestational Age Distribution. Our Results Are Consistent With Research By Ahmed Et Al. That Found No Discernible Differences In Gravidity, Parity, Or Gestational Age Between Cases And Controls.[24]

A Gross Examination Of Placenta In The Control Group Is Normal, With A Mean Central Thickness Of 2.49 Cm, A Mean Number Of Cotyledons Of 16.50, A Mean Weight Of 646.58 Gm, And A Mean Placental Diameter Of 18.10 Cm. It Is Dark Bluish Maroon In Color And Has An Eccentric Attached Cord (46.6%) Or Central (59.3%).

In The Diabetic Group (52.5%), The Oval Form Of The Placenta Was More Common Than In The Normal Control Group (47.5%), Perhaps Due To The Bigger Placentas Associated With Diabetes. The Placenta With Diabetes Had A Dark Blue-Maroon Color, And It Was More Common (53.4%) To Have An Eccentric Cord Attachment Than Control Placentas (46.6%). The More Common Oval Form Of The Larger Diabetic Placentas May Be Responsible For The Eccentric Cord.

The Mean Weight Of The Diabetic Placenta Was Slightly Greater At 646.58g Than The Control Group's 514.59g, A Mean Difference Of 131.99g Which Was Statistically Significant (P-Value < 0.001). With A Mean Central Thickness Of 3.06 Cm For The Diabetes Group And 2.49 Cm For The

Control Group, There Was A Statistically Significant (P-Value < 0.001) Mean Difference Of 5.7 Mm Between The Placentas Of The Two Groups. With A Statistically Significant P-Value (<0.001), The Mean Number Of Cotyledons In The Diabetes Group Was 18.72, While It Was 16.50 In The Control Group. These Measurements Fell Within The Range Of Some Earlier Research Findings. [25,26] The Placentas In The Diabetic Group Exhibited A Significant Accumulation Of Non-Parenchymatous Tissue (Stroma, Glycogen, Lipids, Tissue Fluid Edema And Fibrin) And A Moderate Increase In Parenchymatous (Syncytiovascular) Tissue, Which Could Be Explained By Placental Hyperplasia In Response To Diabetes Which Is In Contrast To Salgi Et Al., The Diabetes Group Showed No Significant Calcification, Hematomas, Thrombosis, Or Infarction On The Mother Placental Surface.[27]

The Mean Weight Of Idms (Infants Of Diabetic Mothers) Increased Significantly In The Diabetic Group, With A Difference Of 280 Grams Above The Control Group. Numerous Studies Have Previously Reported Macrosomic Newborns Among Diabetics. [28,29] Fetal Hyperglycemia Brought On By Maternal Hyperglycemia May Be The Cause Of Macrosomia In Idm. Fetal Hyperinsulinemia Results In Increase In Glycogenesis And Visceral Tissue, Particularly In The Liver, Heart, Muscles, And Viscera, But Not In The Brain, Which Does Not Create Glycogenesis. Additionally, Because Glucose Is Provided As A Source Of Glycerophosphate, Which Is Required For The Esterification Of Free Fatty Acids, The Combination Of Hyperglycemia, Hyperinsulinism, And Increased Free Fatty Acid Availability From The Mother Causes An Acceleration Of Triglyceride Synthesis In Fetal Adipocytes And, As A Result, Obesity In Idm. Similar Conclusions Regarding The Macrosomia Of Idm Had Been Reached By Earlier Research.[30]

A Microscopic Analysis Of The Placentas Of Diabetic Women In Our Study Revealed A Number Of Pathological Abnormalities, Including Decidual Vasculopathy, Thicker Basement Membrane And Villous Immaturity, Chorangiosis, Increased Production Of Syncytial Knots, And Stromal Villous Fibrosis. In Accordance With White's Classification Of Gestational Diabetes Mellitus, There Were No Appreciable Variations In The Microscopic Alterations Of The Placentas Of The Various Groups According To Study By Maksheed M Et Al. [31] Although Verma (2010) [32] Noted That These Placentas Frequently Had Abnormalities, There Was No Consensus On A Consistent Histological Pattern.

**Villous Stromal Fibrosis**

It Is Regarded As Aberrant Since It Is A Morphological Characteristic Of Diminished Villous Perfusion.[33] In The Current Study, 82% Of The Diabetic Placenta Group Had Fibrotic Villi, Whereas The Control Group Did Not Exhibit Any. When Compared To A Normal Placenta, Fox H. Discovered That Diabetic Placentas Have Noticeably More Fibrotic Villi.[34]

**Villous Immaturity**

In Our Study, The Diabetic Placenta Group Exhibited More Villous Immaturity (84%) Compared To 14% In The Control Group. When Compared To A Normal Placenta Group, Laviniagheorman, I.E. Plesea, And V. Gheorma[35] Observed A Substantial Increase In Villous Immaturity In The Diabetic Placenta Group.

Typically, There Is An Increase In The Development Of Vasculosyncytial Membranes And A Faster Differentiation Into Terminal Villi During The Last Trimester Of Pregnancy. However, Growth Factors Like Fetal Insulin Activity Hinder This Final Differentiation In Pregnancies Impaired By Diabetes.[36] Reduced Placental Reserve And Poor Tolerance To Acute Hypoxia Are The Results Of Abnormal Villous Maturation, Which Can Cause Late Intrauterine Fetal Loss.[37]

**Chorangiosis**

Normal Placentas Have A Small Number Of Peripherally Positioned Blood Arteries In Their Villi, And Maternal Blood Is Found In The Intervillous Region. The Thin Placental Barrier In A Healthy Placenta Facilitates The Simple Passage Of Nutrients And Oxygen Between The Blood Of The Fetus

And The Mother.

Diabetic Individuals Exhibit An Increase In Villous Capillaries, With The Majority Of Them Dilated And Crowded Referred As Chorangiosis. In The Current Study, It Was Found That The Diabetic Placenta Group Had Higher Levels Of Chorangiosis (82%), Compared To The Control Group's 20%. In Their Investigation, Natarajan Et Al. Also Discovered Chorangiosis In The Diabetic Group In Contrast To The Normal Placental Group.[38]

**Thickening Of Basement Membrane**

In Comparison To The Control Group, Which Had No Basement Membrane Thickening, 86% Of The Placentas Of Diabetes Women In Our Study Had A Noticeable Thickening Of The Villi's Basement Membranewhen Compared To A Normal Placenta Group, Tewari V., Tewari A., And Bharadwaj N. Discovered That The Thickness Of The Basement Membrane Significantly Increases In The Diabetes Placenta Group.[39]

**Fibrinoid Necrosis**

In Our Investigation, We Found That 86% Of The Case Group Had Fibrinoid Necrosis Of The Maternal And Fetal Blood Vessels, Whereas The Control Group Did Not Have Any. Fibrinoid Necrosis Was Found To Be Considerably Higher In The Diabetic Group (6.70±2.14) As Compared To A Mean Value Of 2.25±1.66 In The Control Group, According To Pankaj Saini Et Al. [40] Similar Results Were Also Reported By Treesh Sa, Augustine Et Al., Khair Ns Et Al., And Kalla Raviteja Et Al. In Their Investigations.

**Villous Edema**

In The Current Study, We Found That In The Diabetes Group, 80% Of Placentas Had Villous Edema, Whereas In The Control Group, There Was No Villous Edema. Wasserman L, Schlesinger H, Et Al. Analyzed These Kinds Of Alterations With A Focused Distribution On The Distal Villi In Most Of The Patients.[41] The Formation Of Aberrant Deposits Of Mucopolysaccharides In The Villous Stroma Was Found To Be The Likely Cause Of The Genuine Villous Edema In The Placenta Of Diabetes Mothers.

**Syncytial Knot**

Synovial Knots Are Groups Of Very Small, Strongly Stained Nuclei That Stick Out From The Villous Surface And Into The Intervillous Area. According To The Current Study, The Control Group Had 34% Syncytial Knots, Whereas The Group Of Placentas With Diabetes Had 100% More Syncytial Knots. Augstine Et Al. Also Reported The Same Conclusion.

The Diabetic Group In The Current Study Had 68% Calcification, Which Is Significantly Higher Than 14% Seen In The Control Group. Similarly, In The Gdm Group, 82% Of Patients Had Maternal Decidual Vasculopathy. The P-Value < 0.001 Indicates A Substantial Correlation Between The Aforementioned Histological Alterations And The Gdm Placenta, Which Could Have A Negative Impact On The Newborn's Prognosis.

46% Of Patients In The Diabetes Group And 36% In The Control Group Had Delivered Via Caesarean Section. The Mode Of Delivery Differs In Both Groups, But It Is Not Statistically Significant (P = 0.309). Our Study's Findings Are In Line With Those Of Several Other Research, Including One By Yogev Et Al. (30%) And One By Evers Et Al. (44%). [42,43] The Most Common Indications For Lscs In The Diabetic Group Were Cpd (12%), And Prior Lscs (26%). Fetal Distress (6%), As Well As Prior Cs (22%), Were Common Indications For Lscs In The Control Group.

**Neonatal Outcome**

Birth Weight Comparison Between The Groups Revealed That Babies Born To Diabetic Mothers Typically Weighed 3.50 Kg, Compared To Controls' 3.22 Kg, A Mean Difference Of 280 Grams That Is Statistically Significant (P-Value = 0.001). There Is A Strong Correlation Between Increased Birth

Weight And Gdm.

68% Of Gdm Patients Received A Diet Plan, 22% Received A Diet Plan Combined With Insulin Medication, And 10% Did Not Receive Any Treatment At All.

32% Of Babies In The Gdm Group Had Hypoglycemia, 42% Had Macrosomia, And 40% Had Hypocalcaemia. The Case Group Exhibits Polycythemia In 6% Of Cases And Hyperbilirubinemia In 36% Of Cases.

In Their Investigation, Jacques Et Al. Discovered That The Incidence Of Hypoglycemia Was 3.2%. According To Evers Et Al., The Incidence Of Hyperbilirubinemia Was 25% And Macrosomia Was 45%.(176) A Study By Priyanka Et Al. Revealed That The Incidence Of Hyperbilirubinemia Was 12%, Hypoglycemia Was 9%, And Macrosomia Was 18%.[44]

The Comparison Of Fetal Outcomes In The Various Treatment Groups Showed That Patients Who Received The Diet Plan Had A Greater Incidence Of Hypoglycemia (41.2%) Than Patients Who Received The Diet Plan With Insulin Treatment (18.2%), With A P-Value Of 0.002. Mothers Who Followed A Diet Plan Experienced A Similar Incidence Of Macrosomia (38.2%) As Those Who Followed A Diet Plan With Insulin Treatment (45.5%, P = 0.032). There Was A Strong Correlation (P Value<0.05) Between Gdm And The Incidence Of Hypoglycemia And Macrosomia In Newborns. The Incidence Of Hypocalcemia Is 35.3% In The Group That Followed The Diet Plan And 54.5% In The Group That Followed The Diet Plan Plus Insulin Treatment (P = 0.231). Only The Diet Plan With Insulin Treatment Group (P = 0.093, 9.1%) Exhibits Polycythemia.

The Diet Plan Group Had A 29.4% Incidence Of Hyperbilirubinemia, While The Diet Plan With Insulin Treatment Group Had A 54.5% Incidence (P = 0.321). We Found No Significant Correlation Between Gdm And The Development Of Hypocalcemia, Polycythemia, Or Hyperbilirubinemia, With A P-Value Of >0.05. In Mothers Who Had Not Had Any Treatment, The Incidence Of Macrosomia Was 60%, Hypocalcemia Was 40%, Polycythemia Was 40%, And Hyperbilirubinemia Was 40%.

## Conclusion

This Study Clearly Shows That Mothers Who Did Not Receive Any Therapy Had A Higher Percentage Of Unfavorable Neonatal Outcomes Than Patients Who Followed Diet Plans Or Diet Plans With Insulin Medication. Developing Guidelines For Prevention And Treatment To Improve Perinatal Outcomes Will Be Easier With A Better Understanding Of Placental Etiology.

## References

1. American Diabetic Association: Report Of The Expert Committee On The Diagnosis And Classification Of Gestational Diabetes Mellitus. Diabetes Care 2003;26(1 Suppl):103-5.
2. Siddiqui S, Waghdhare S, Panda M, Sinha S, Singh P, Dubey S, Et Al. Regional Prevalence Of Gestational Diabetes Mellitus In North India. J Diabetol 2019;10(1):25-8.
3. Gestational Diabetes Mellitus. Current Guidelines For Diagnosis And Management Medicine Update 2010;20.
4. Kayal A, Anjana Rm, Mohan V. Gestational Diabetes-An Update From India. Diabetes Voice 2013;58(2):32-4.
5. Patric Mc. Diabetes Mellitus. In Reproductive Endocrinology, Surgery And Technology. Philadelphia Usa: Lippincott 1996.
6. Gauster M, Desoye G, Totsch M, Hiden U. The Placenta And Gestational DiabetesMellitus. Curr Diab Rep 2012;12(1):16-23.
7. Calderon Im. Morphometric Study Of Placental Villi And Vessels In Women With Mild Hyperglycaemia Or Gestational Or Overt Diabetes. Diabetes Res Clin Pract 2007;78:65-71.
8. Jones Cj, Fox H. Syncytial Knots And Intervillous Bridges In The Human Placenta: An Ultra-Structural Study. J Anat 1977;124:275-86.
9. Saddler Yw. Placenta And Fetal Membrane. Longman's Medical Embryology. Usa: Lippincott Williams And Wilkins 2004.
10. Singer Db. The Placenta In Pregnancies Complicated By Diabetes Mellitus. Perspect Peditr

Pathol 1984;8(3):199-212.
11. Hanson Ui, Persson B. Outcomes Of Pregnancies Complicated By Type 1 Insulin – Dependent Diabetes In Sweden: Acute Pregnancies Complications, Neonatal Mortality And Morbidities. Am J Perinatol 1993;10(104):330-3.
12. Langer O, Yogev Y. Gestational Diabetes: The Consequences Of Not Treating. Am J Obstet Gynecol 2005;192:989-97
13. Gabbay-Benziv R, Baschat Aa. Gestational Diabetes As One Of The "Great Obstetrical Syndromes"- The Maternal, Placental And Fetal Dialogue. Best Pract Res Clin Obstret Gynecol 2015;29(2):150-5.
14. Honda M, Toyoda C, Nakabayashi M, Omori Y. Quantitative Investigation Of Placenta Terminal Villi In Maternal Diabetes Mellitus By Scanning And Transmission Electron Microscopy. Tohoku J Exp Med 1992;167(4):247-57.
15. Laurini Rn, Visser Gh, Van Ballegooie E, Schoots Cj. Morphological Findings In Placenta Of Insulin Dependent Diabetic Patients Treated With Continuous Subcutaneous Insulin Infusion (Csii). Placenta 1987;8(2):153-65.
16. Thomsen K. Defective Development Of Young Placenta Villi. Arch Gynacol 1955;185(6):807-33.
17. Burstein R, Handler Fp, Soule Sd, Blumenthal Ht. Histogenesis Of Degenerative Processes In The Normal Mature Placenta. Am J Obstet Gynecol 1956;72(2):332-42.
18. Thomsen. Findings On Placental Morphology In Diabetes Mellitus. Acta Endocrinol(Copenh) 1958;29:602-14.
19. Stoj F, Scehumhmann Ra. Morphometric Investigation Of Terminal Villi Of DiabeticPlacenta In A Relation To The White's Classification Of Diabetes Mellitus. J Pernat Med 1987;15:193-8.
20. Younes B, Baez-Giangreco A, Al-Nuaim L, Al-Hakeem A, Talib Za. Basement Membrane Thickening In The Placenta From Diabetic Women. Pathol Int 1996;46(2):100-4.
21. Al-Okail Ms, Al-Attas Os. Histological Changes In Placenta Syncytiotrophoblast Of Poorly Controlled Gestational Diabetes Patients. Endocr J 1994;41(4):355-60.
22. Kheir Ae, Berair R, Gulfan Ig, Karrar Mz, Mohammed Za. Morbidity And Mortality Amongst Infants Of Diabetic Mothers Admitted Into Soba University Hospital, Khartoum, Sudan. Sudanese Journal Of Paediatrics 2012;12(1):49-55.
23. Farooq Mu, Ayaz A, Bahoo La, Ahmad I. Maternal And Neonatal Outcomes In Gestational Diabetes Mellitus. Int J Endocrinol Metab 2007;5(3):109-15.
24. Elshennawy Tm, Halima Aa. Effect Of Gestational Diabetes On Gross Morphology, Histology And HistochemistryOf Human Placenta. Endocrinol Metab Synd 2016;5(1):1-13.
25. Wilczyński J, Podciechowski L, Krekora M, Wenerski J, Czichos E, Kulig A, Et Al. Macroscopic Estimation Of The Placenta Using A Morphometric Grid. Part I: Pre-Pregnancy And Post-Pregnancy Diabetes Mellitus. Ginekologia Polska 1998;69(12):974-81.
26. Evers Im, Nikkels Pg, Sikkema Jm, Visser Gh. Placental Pathology In Women With Type 1 Diabetes And In A Control Group With Normal And Large-For-Gestational-Age Infants. Placenta 2003;24(8-9):819-25.
27. Salge Ak, Rocha Km, Xavier Rm, Ramalho Ws, Rocha Él, Guimarães Jv, Et Al. Macroscopic Placental Changes Associated With Fetal And Maternal Events In Diabetes Mellitus. Clinics 2012;67(10):1203-8.
28. North Jr Af, Mazumdar S, Logrillo Vm. Birth Weight, Gestational Age, And Perinatal Deaths In 5,471 Infants Of Diabetic Mothers. J Pediatrics 1977;90(3):444-7.
29. Gabbe Sg, Mestman Jh, Freeman Rk, Goebelsmann Ut, Lowensohn Ri, Nochimson D, Et Al. Management And Outcome Of Pregnancy In Diabetes Mellitus, Classes B To R. Am J Obstet Gynecol 1977;129(7):723-9.
30. Kalhan Sc, Savin Sm, Adam Pa. Attenuated Glucose Production Rate In Newborn Infants Of Insulin-Dependent Diabetic Mothers. N Engl J Med 1977;296(7):375-6.
31. Makhseed M, Musini Vm, Ahmed Ma, Al-Harmi J. Placental Pathology In Relation To The

White's Classification Of Diabetes Mellitus. Arch Gynecol Obstet 2002;266:136-40.
32. Verma R, Mishra S, Kaul Jm. Cellular Changes In The Placenta In Pregnancies Complicated With Diabetes. Int J Morphol 2010;28(1):259-64.
33. Fox H. Fibrosis Of Placental Villi. J Pathol Bacteriol 1968;95(2):573-9.
34. Fox H. The Significance Of Villous Syncytial Knots In The Human Placenta. Bjog: An International Journal Of Obstetrics & Gynaecology 1965;72(3):347-55.
35. Gheorman L, Pleşea Ie, Gheorman V. Histopathological Considerations Of Placenta In Pregnancy With Diabetes. Rom J Morphol Embryol 2012;53(2):329-6.
36. Redline Rw. Distal Villous Immaturity. Mini Symphosium: Placental And Trophoblastic Pathology. Diagnost Histopathol 2012;18(5):189-94.
37. Paciencia M, Dolley P, Jeanne-Pasquier C, Jacob B, Sadfi A, Leseigneur P, Et Al. Acute Placental Dysfunction By Villous-Maturation Defect And Late-FetalMortality. J Gynecol Obstet Biol Reprod 2008;37(6):602-7.
38. Natarajan L, Maheswari Gu. Gestational Hyperglycemia On Diet And Medication: Impact On Placental Pathology And Pregnancy Outcomes. Int J Reprod Contracept Obstet Gynecol 2019;8:3350-6.
39. Tewari V, Tewari A, Bhardwaj N. Histological And Histochemical Changes In Placenta Of Diabetic Pregnant Females And Its Comparison With Normal Placenta. Asian Pacific Journal Of Tropical Disease 2011;1(1):1-4.
40. Saini P, Pankaj Jp, Jain A, Agarwal Gc. Effect Of Gestational Diabetes Mellitus On Gross Morphology Of Placenta: A Comparative Study. Int J Anat Res 2015;3(1):889-894.
41. Wasserman L, Shlesinger H, Abramovici A, Goldman Ja, Allalouf D. Glycosaminoglycan Patterns In Diabetic And Toxemic Term Placentas. Am J Obstet Gynecol 1980;138(7):769-73.
42. Evers Im, De Valk Hw, Visser Gh. Risk Of Complications Of Pregnancy In Women With Diabetes: Nationwide Prospective Study In Netherland. Bmj 2004;328(7445):915-9.
43. Yogev Y, Xenakis Em, Langer O. The Association Between Preeclampsia And The Severity Of Gestational Diabetes: The Impact Of Glycemic Control. Am J Obstet Gynecol 2004;191(5):1655-60.
44. Kalra P, Kachhwaha Cp, Singh Hv. Prevalence Of Gestational Diabetes Mellitus And Its Outcome In Western Rajasthan. Indian J E Endocrinol Metab 2013;17(4):677-80.